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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
08/764,110	12/06/96	CHEN	Y

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EXAMINER	
BERCH, M	
ART UNIT	**PAPER NUMBER**
1624	21

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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.	Applicant(s)
08/764,110	Chen
Examiner	**Group Art Unit**
Mark L. Berch	1624

☒ Responsive to communication(s) filed on *Mar 23, 2000* .

☒ This action is **FINAL**.

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire *3* month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claims

☒ Claim(s) *2-4, 8-10, 12-14, 18, and 20-24* is/are pending in the application.

Of the above, claim(s) _____ is/are withdrawn from consideration.

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) *2-4, 8-10, 12-14, 18, and 20-24* is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐approved ☐disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☐ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____ .

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____ .

☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☐ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☐ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

--- SEE OFFICE ACTION ON THE FOLLOWING PAGES ---

DETAILED ACTION

Continued Prosecution Application

The request filed on 3/23/00 for a Continued Prosecution Application (CPA) under 37 CFR 1.53(d) based on parent Application No. 08/764,110 is acceptable and a CPA has been established. An action on the CPA follows. It is noted that there was no request for entry of the unentered amendment filed 9/7/99.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 2-4, 8-10, 12-14, 18, 20-24 are rejected under 35 U.S.C. 112, paragraphs 1 and 2, as the claimed invention is not described, or is not described in such full, clear, and exact terms as to enable any person skilled in the art to make and use the same, and/or failing to particularly point out and distinctly claim the subject matter which applicant regards as his invention. Specifically:

1. The term "pyschosocial dwarfism" is indefinite. Simply finding definitions for the two individual words does not make the term itself definite. The term is included in a

list of medical conditions to be treated; applicants have presented no evidence that this is a defined medical condition, so that one of ordinary skill in the art could distinguish this dwarfism from other dwarfisms. (paragraph 2)

2. The term (a) language is vague. Its scope is unknown. Determining whether a given disease responds or does not respond to such antagonism will surely involve undue experimentation. Suppose that a given CRF Antagonist X when administered to a patient with Disease D does not obtain a response. Does one then conclude that Disease D does not fall within this claim? Keep in mind that:

A. It may be that the next patient will respond. It is quite common for pharmaceuticals to work only with some people, not all. Thus, how many need to be tested?

B. It may be that the wrong dosage or dosage regimen was employed. It is quite common for pharmaceuticals to work at one dosage, but not at another which is significantly higher or lower. Furthermore, the dosage regimen may be vital --- should the drug be given e.g. once a day, or four times in divided dosages? Thus, how many dosages and dosage regimens must be tried before one is certain that this pharmaceutical won't affect Disease D?

C. It may be that X simply isn't potent enough for Disease D, but that another antagonist Y is potent enough, so that D really does fall within the claim. Thus, how many different antagonists must be tried before one concludes that D doesn't fall within the claim?

D. Conversely, if D responds to Y but not to X, can one really conclude that D falls within the claim? It may be that the X result is giving the accurate answer, and that the success of Y arises from some other unknown property which Y is capable of. Thus, when mixed results are obtained, how many more pharmaceuticals need be tested?

E. Finally, suppose that X really will work, but only when combined with Z. There are for example, agents in the antiviral and anticancer technology which are not themselves effective, but the disease will respond when the agents are combined with something else.

F. In addition, literally speaking, any disorder can be treated with any drug, although the treatment might not be successful. Assuming that "successful treatment" is what is intended, what criterion is to be used? If one person in 10 responds to a given drug, does that mean that the disease is treatable? One in 100? 1,000? 10,000?

As a result, determining the true scope of the claim will involve extensive and potentially open-ended research. Without it, one skilled in the art cannot determine the actual scope of the claim. Indeed, it is not clear that there is any disorder that one can state with confidence does not fall within the claim. Hence, the claim is indefinite. Applicants' previous traverse on this point is unpersuasive. The examiner is not requiring "applicant to test each and every embodiment of the invention". The examiner isn't suggesting that applicant test anything here. The examiner is doing

the reverse --- noting that this sort of claim language forces on the public a potentially unlimited series of tests to determine the scope of the claim. The actual scope of term (a) is simply not known at present; for all we know, every disease --- or none --- fall into this category. Determining the actual scope of this term, at this point in time, requires extensive and fundamental research, which is improper to place on the public.. The burden is on applicant to particularly point out and distinctly claim the scope of the invention. Except for experimentation of a routine nature, this burden cannot be placed on the public, and that is exactly what this language does. For the reasons set forth in the lettered points above, determining for sure which diseases do and which do not fall within the ambit of this language is a difficult undertaking, one that is not impossible but one that involves undue experimentation. Applicants had mentioned animal tests. These are useful, but an animal test does not determine whether a disease falls within this category; it merely identifies which drugs might be successful for a given disease. The question here is the meaning of the claim language, not whether any given compound will be effective against any given disease. (paragraph 2)

3.The replacement of the term "thioalkyl" with "alkylthio" is clearly new matter. There is no way of telling whether that term or "mercaptoalkyl" is what was originally intended; applicants have simply made a determination after the filing date. Although the original terms was clearly defective, both "alkylthio"and "mercaptoalkyl" are reasonable possibilities as to what was originally intended. Applicants pointed to the

fact that in the Miller patent, thioalkyl clearly meant alkylthio. Yes, that is one reasonable meaning for the term, but its isn't the only one. In Miller, it was indeed clear what meaning was actually intended because of the example. But no such fact situation occurs here. There is no way of knowing whether in this case it meant alkylthio or mercaptoalkyl. Applicants have made an arbitrary selection. Applicants have not shown that one of ordinary skill in the art would have known which of two possible readings to take in this case. (paragraph 2)

4. The optionally substituted pyrimidyl choice for R^5 lacks enablement; this term did not exist in the specification's definition of R^5 as originally filed. Applicants pointed to page 9, lines 9-15. First, this material is broader than has been put into Claim 18 or the specification. It is limited to 2 substituents (not up to four as is seen in Claim 18 and page 4), and the substituents are drawn from a much smaller list. Thus, the specification and claims now cover amino pyrimidinyl, or trimethylpyrimidinyl, terms that are not covered by the page 9 material. In addition, the subject matter of the paragraph is limited in other ways. It has a much narrower definition of R^3 than does Claim 18 or the specification. Thus, a compound with R^3 as CN is now in Claim 18 or the specification, but is not on page 9. The same is true of other variables such as R^4. Moreover, even material which is described lacks enablement. The utility is tied to the formulae I, II, and III as defined. Additional material outside that was invented by applicants, but isn't covered by the utility umbrella. (paragraph 1)

5. Applicants have added CF_3 to the R^4 definition. This has the same problems. Just an example, page 9 does not provide for a compound with R^3 as CN where R^4 is CF_3, but now the claim does. (paragraph 1)

6. The same is true for the three new choices for R^{12}. For example, Claim 18 now has fluoronaphthyl as a choice for R^5; no such thing occurs on page 9. (paragraph 1)

7. The provision for the optional multiple bond for R^5 now inserted into Claim 18 lacks enablement and description. Applicants point to page 14, lines 3-5, but that does not cover R^5. (paragraph 1)

8. The inclusion of "inflammatory diseases" is clearly new matter and lacking in description. Applicants pointed to page 1, lines 29-34, but that is talking about the prior art. It does not say that the claimed compounds are effective for this. The specification hasn't even started to talk about the claimed compounds yet. This is all background. Further the sentence is actually referring only to "stress-related illnesses" Thus, inflammatory diseases that don't arise from stress wouldn't be included in that sentence, but are now in the claims. (paragraph 1)

9. Further, the inclusion of this term now means that other terms appear to be superfluous. Thus, the very next term is an inflammatory disease, and hence is already included by the new term. (paragraph 2)

10. An HIV infection is not a disorder; it needs its own category. A disorder is a disturbance in the regular and natural functions of either body or mind. An infection is not part of the regular and natural functions of either body or mind and hence it is

not a disturbance in it. Of course, an infection can trigger a disorder, such as a fever or damage to the immune system. Thus, the fever is the disorder, not the infection. The infection is the cause of the disorder, but not the disorder itself. Not all bodily problems are disorders. Things like infections and wounds (e.g. bee sting) can subsequently cause a disorder, but are not themselves a disorder. (paragraph 2)

11. Claim 22 lacks description. Even a negative limitation requires description, *Ex Parte Graselli,* 231 USPQ 393. (paragraph 1)

12.Further, it is unclear how it operates. Is a specie excluded if it meets any of the conditions or only if it meets all three of the conditions. (paragraph 2)

Claims 2-4, 8-10, 12-14, 18, 20-24 are rejected as being drawn to an improper Markush groups for reasons set forth previously in the paper of 7/25/97. Limiting the claims to pyrrolopyrimidines will overcome the rejection. Applicants' traverse on this point was unpersuasive. Each provides a different nucleus. The fact that each nucleus has 9 atoms and two rings does not change the fact that these are different. Purines and imidazopyridines are each a different heterocyclic nucleus. Contrary to what applicants stated, the classification is indeed different; e.g. imidazopyridines are in Class 546, not 544.

Claims 20-21 and 23-24 are rejected, 35 USC 112, paragraph 1, for lack of enablement for such scope.

No medicinal even known to man has ever been capable of treating such a staggering scope of disorders. To get a single compound, let alone a genus of

billions, to be effective against such a vast array of disorders has been beyond the reach of medicine. The failure to achieve such a goal in the past places the burden on applicants to show that their compounds really can accomplish this, Cf In re Ferens, 163 USPQ 609. Indeed, a scope so broad in effect forces the public to unduly experiment to determine the actual utility. This is an impermissible burden; see In re Schmidt 153 USPQ 640, which has a much smaller disclosure than is seen here. The Supreme Court stated in *Brenner v. Manson*, 383 U.S. 519, 536, 148 USPQ 689, 696 (1966) "a patent is not a hunting license. It is not a reward for the search, but compensation for its successful conclusion." This specification, with its staggering array of utilities and mechanisms, is no more than an invitation for the public to figure out for themselves how to use these compounds.

It is allegedly effective against a huge variety of psychological disorders (e.g. recurrent and much more), inflammatory disorders (e.g. allergies), a variety of neurodegenerative disorders (e.g. AD), all chemical dependencies regardless of type, CNS disorders (Stroke) and developmental disorders (dwarfism). It is claimed to be used in good number of the body's basic systems, such as cardiovascular (hypertension, tachycardia), gastrointestinal (IBS, spastic colon, ulcers), joints (rheumatoid arthritis), immune (immune suppression), muscular (muscular spasms), excretory (urinary incontinence) etc. It is effective against disorders normally thought of a untreatable, such as mutiinfarct dementia. Applicants' genus of billions

of compounds has such a wide range of action as to be a general panacea. However, such a generalized panacea is not deemed enabled, *In re Citron*, 129 USPQ 520.

This is particularly true for something like cancer. The claim sets forth the treatment of cancer generally. However, there never has been a compound capable of treating cancer generally. Applicants argue that there are compounds that treat a range of cancers. This is true, but no one has ever been able to figure out how to get a compound to be effective against cancer generally, or even a majority of cancers. Even the most broadly effective antitumor agents are only effective against a small fraction of the vast number of different cancers known. Despite the understanding, as applicants state, that chemotherapeutic agents destroy malignant cells without substantially interfering with the growth of normal cells, it is beyond the skill of oncologists today to get an agent to be effective against cancers generally.

Much the same is true, for example, for "chemical dependencies and addictions". The notion that a compound could be effective against chemical dependencies in general is absolutely contrary to our current understanding of how chemical dependencies operate. There is not, and probably never will be, a pharmacological treatment for "drug addiction" generally. That is because "drug addiction" is not a single disease or cluster of related disorders, but in fact, a collection with relatively little in common. Addiction to barbiturates, alcohol, cocaine, opiates, amphetamines, benzodiazepines, nicotine, etc all involve different parts of the CNS system; different receptors in the body. For example, cocaine binds at the

dopamine reuptake transmitter. Heroin addiction, for example, arises from binding at the opiate receptors, cigarette addiction from some interaction at the nicotinic acid receptors, many tranquilizers involve the benzodiazepine receptor, alcohol involves yet another system, etc. All attempts to find an pharmaceutical to treat chemical addictions generally have thus failed.

Applicants list AIDS. All successful treatments of AIDS have involved antiviral agents. No one has ever been able to get other attempts to work. This shows that the skill level in this art is not high enough to get other methods to work. Similarly with the treatment of AD. Getting agents to be effective against AD has proven extremely difficult. Despite extraordinary efforts with a variety of agents in this area, only two pharmaceuticals have been made to work, both acetylcholinesterase antagonists, a property that these compounds are not disclosed to have. No one has been able to figure out how to get CRF regulators to be effective against AD, which is evidence of the low skill level in this art relative to the difficulty of the task.

To further rebut applicants' arguments, Chalmers is cited as an example of the skill level in this art as of 1996. Note especially pages 171-172, which deal with "Therapeutic Strategies". Three things are of particular relevance:

1. AD and eating disorders, especially obesity are associated with abnormally low levels of CRF. Therefore, administration of CFR antagonists, if they had any effect, would be expected to make matters worse.

2. Stroke appears in the claim. Chalmers says (page 170) that "there are no effective neuroprotective agents that can clinically ameliorate the effect of stroke in humans." This is evidence that, despite decades of effort to treat stroke, that the skill level in the art is just too low relative to the task to figure how to get a pharmaceutical to do this.

3. The full paragraph on page 171 lists several general strategies for high-CRF disorders. It is clear from the wording, however, that as of 1996, it had not been determined to how or even whether this proposed strategy was going to be made to work. The paragraph speaks about possible progress in the future, such as how to target the drug to a particular tissue or region. Progress is talked about as coming in the future, to "provide potential treatment for a number of disease states." The article makes no mention at all of anyone having figured out as of 1996 how to actually do this. The concluding remarks on page 172 state explicitly that it is "future investigation of multiple CRF subtypes [a consideration of which doesn't even appear in this specification] ... will also provide a basis for rational drug design for the treatment of disease states that are associated with abnormal CRF levels." This is clear evidence that as of 1996, such a utility was not enabled.

A second piece of evidence is the Stratakis reference, showing state of the art in 1997. In 6 pages of discussion of CRF, there is only a single sentence devoted to use of CRF antagonists. It is clear from the wording that such efficacy is in the future --- these "might prove useful". Also note that the following sentence adds atypical

depression, chronic fatigue/fibromyalgia and autoimmune disorders to the list of disorders which have CRF levels too low, which CFR antagonists would be expected, if anything , to make worse.

Applicants cited *Cross vs Iizuka*, 224 USPQ 739. But the facts were very different there. Cross had a specific enzyme considered to be useful per se for the treatment of asthma. The sole question there was whether any utility had been established for the compounds. Here that question doesn't arise because the compounds are not under rejection; the question is that of the vast scope recited in the claims. The same is true of *Nelson v. Bowler*, 206 USPQ 881. Moreover, in that case there was direct testing, employing a test that had an excellent track record of 5 years, which showed that the compound was directly useful in regulating blood pressure. This established a utility, which was all that he needed to have the compounds enabled. The scope issue simply did not arise in that case.

In discussing Chalmers and Stratakis, applicants stated that "...increases in CRF are related to ..." followed by large lists of disorders. But a mere relationship does not mean that one skilled in the art would be able to use the claimed compounds without undue experimentation. If they could, all diseases would long ago, be treatable, since virtually every disorder has been related to one or more chemical, receptor, etc in the body. Thus, for example applicants state that Chalmers "indicates a connection between CRF" and Alzheimer's Disease. Dozens if not hundreds of proteins, cytokines, hormones, enzymes, neurotransmitters, etc have been found to be

connected in some way to Alzheimer's Disease. But for virtually none of them have those skilled in the art been able to figure out how to use this connection. It is often very difficult to even determine if the chemical (usually, changes in the level of the chemical) is a cause or an effect of a disorder. If the change is an effect of the disease, then one would not ordinarily expect that changing the level of the chemical would have any practical benefit. But even if a chemical is clearly established as a cause, figuring out how to get something which effects it to treat the disease can be extraordinarily difficult. Thus, it is well established that levels of a certain neurotransmitter are far too low in the brains of Alzheimer's Disease patients. The Acetylcholinesterase enzyme destroys this enzyme, and thus an Acetylcholinesterase inhibitor would logically be expected to be effective. Such inhibitors have been known for over fifty years; hundreds and probably thousands have been identified. A callosal amount of effort has produced only two such compounds effective in any way, evidence of the extreme difficulty in going from information about the fact that a compound has a "connection" to a disorder to actually treating the disorder.

Thus, while applicants said that "CRF antagonism is relevant to many pharmacological uses...", that does not mean that such relevance can be translated into treatment of disorders without undue experimentation. Thus, for example, the fact that there is a "connection between CRF antagonism and stroke" does not mean that one skilled in the art could without undue experimentation figure out how to get these compounds (billions of compounds) to be effective against stroke. Stroke

represents one of the most intractable medical challenges. Stroke is estimated to cause about 15% of deaths, behind only heart disease and cancer. Even those who survive normally suffer from persistent damage, including motor and speech disturbances and/or convulsions. Despite a tremendous effort to resolve these problems, cerebrovascular therapy as so far been limited to trying to prevent further damage in areas on the margins of the ischemic focus, thus trying to maintain adequate perfusion in remaining intact areas, and thereby limit progressive infarction. This is generally done surgically. Standard pharmaceutical treatment, such as antiarrhythmics and antithrombotics don't get at the cause of the stroke or the damage caused, but are mostly done to insure adequate cardiac functioning.

Effective acute drug treatment of the stroke itself has so far proved to be beyond the reach of medical science. Major efforts have certainly been pressed in the are of neuroprotective therapeutics. Those studied have included use of Ca antagonists such as Levemopamil and flunarizine, to suppress neuronal calcium influx; NMDA antagonists (both competitive, such as APV and CPP, and non-competitive such as chlorpromazine, ifenprodil and Mg salts) as well as AMPA and kainate antagonists to block post-ischemic receptor-operated calcium channels; attempts to block arachidonic acid cascade or elimination of its metabolic products with agents such as lipogenase inhibitors and thromboxane; use of free oxygen radical scavengers such as superoxide dismutase, alpha-tocopherol, or allopurinol to inhibit the lipid peroxidation that damages cell membranes, which may indirectly help

prevent intracellular calcium overload; anti-edema agents such as corticosteroids; use of 5-HT_{1A} receptor agonists to suppress 5-HT concentrations in the hippocampal extracellular space; use of CRF receptor antagonists to inhibit excitotoxic brain damage; use of serotonin 1A agonists such as ipsapirone, or adenosine modulators such as vinpocetine, to stimulate adenosine, which may act as a protective agent by hyperpolarizing the postsynaptic neuron; use of platelet aggregation inhibitors such as prostacycline and ticlopidine, and other approaches as well.

Despite this vast outpouring of research, the skill level in this art is sufficiently low relative to the difficulty of the task that obtaining treatment of stroke was, as of the filing date, not yet possible. Hence, accomplishing such a goal involves more than routine experimentation. As evidence for this, there was cited Chalmers which states flatly on page 170 that, "At present, there are no effective neuroprotective agents that can clinically ameliorate the effects of stroke in humans." As an example of this massive effort, Pentoxifylline has been one of the most intensely studied, with literally dozens of studies published on its properties. It appears to have a wide variety of effects on leucocytes, erythrocytes, neutrophils, plasma fibrinogen levels. These result in a wide ranging ability to increase blood flow, resulting in effectiveness in some vascular disorders, especially intermittent claudication. Yet, it is still unclear whether this drug can be made to work against stroke. Research with different administration methods, or different subcategories of stroke may well result in the discovery of how to get this drug to work, but the slowness and difficulty of this

research shows clearly that this involves undue, not routine experimentation. Applicants' compounds have been subjected to no such study.

Applicants mentioned cancer but their response really doesn't get to these issues mentioned above. Yes, there is a "connection" between CRF and the immune system, and between the immune system and cancer, but that is a far distance from establishing that CRF inhibitors can treat cancer generally, a feat that has never been accomplished in the history of oncology.

Next, applicants mentioned chemical dependencies and addictions and present a reference on REVIA. Those skilled in the art have found a way to use this for opiate dependency, which is not surprising since that drug binds to the opiate receptor. It is not seen how this is of any relevance here. Applicants' compounds are not disclosed to bind to the opiate receptor, nor have any compounds which don't bind to the opiate receptor ever been made effective for treating opiate dependence. Further, applicants argument does not get at the problem that the claim covers treatment of addictions generally. For reasons set forth above, since these have so little in common, one would not expect a chemical to treat the broad range of addictions.

A similar problem exists for "drug and alcohol withdrawal symptoms." Applicants cite a reference on Tranxene, which is indicated for the relief of alcohol withdrawal. But this is a standard benzodiazepine antianxiety drug; it acts by binding to the BZ receptor. Why should a CRF antagonist be expected to do what a BZ

antagonist can do? Further, there is again a question of scope. After all, Tranxene could probably not be used for withdrawal from benzodiazepines!

Applicants are correct that anorexia nervosa should not have been included in the list of the Final Rejection of 3/26/98, page 4, item 1. However, the examiner's argument stills stands with regard to obesity and Alzheimer's Disease, as these are associated with abnormally low, not abnormally high levels of CRF, as Chalmers states.

The claims now have HIV infections, but the previous reasoning concerning AIDS applies just as well here. No one has ever been able to treat any HIV infection -- - indeed, any viral infection at all, except by means of an antiviral, something that disrupts the operation of the virus itself. There is no reason to think that a CRF antagonist could possibly do this, since no virus actually uses this hormone for any aspect of its replication, infectivity, etc.

Applicants have cited Owens, a large review article on CRF. As this is an older (1991) reference, it cannot supply information about the state of the art at the time of the invention. However, it is instructive to note the last two sentences of the text. This states explicitly that the research necessary to understand "basic CRF physiology" remains to be done, and that "pharmacological agents" are only "possibilities", not realities. Lyons presents a hypotheses that needs further support (see last sentence). Suemaru (which is reviewed only for the Abstract; the rest is in

Chinese) Strijbos and Suda are all basic research papers, not dealing with the treatment of disease. Fackelmann is noted but does not mention CRF.

All claims are drawn to the same invention claimed in the parent application prior to the filing of this Continued Prosecution Application under 37 CFR 1.53(d) and could have been finally rejected on the grounds and art of record in the next Office action. Accordingly, THIS ACTION IS MADE FINAL even though it is a first action after the filing under 37 CFR 1.53(d). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the Examiner should be directed to Mark L. Berch whose telephone number is 703-308-4718.

Mark L. Berch

Primary Examiner

Group 1610 - Art Unit 1624

April 7, 2000